

Is the Relationship between Race and CPAP Adherence Mediated by Sleep Duration?

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Abstract

Study Objectives: Black race has been associated with decreased CPAP adherence. Short sleep duration, long sleep latency and insomnia complaints may affect CPAP adherence as they impact sleep and opportunity to use CPAP. We assessed whether self-reported sleep measures were associated with CPAP adherence and if racial variations in these sleep characteristics may explain racial differences in CPAP adherence.

Design: Analysis of data from a randomized controlled trial (HomePAP), which investigated home vs. lab based diagnosis and treatment of OSA

Setting: Seven AASM-accredited sleep centers in 5 US cities

Patients or Participants: Enrolled subjects (N=191) with AHI \geq 15 and sleepiness (ESS $>$ 12)

Interventions: N/A

Measurements and Results: Multivariable regression was used to assess if subjective sleep measures and symptoms predicted 3 month CPAP use. Mediation analysis was used to assess if sleep measures mediated the association of race with CPAP adherence. Black participants reported shorter sleep duration and longer sleep latency at baseline than white and Hispanic participants. Shorter sleep duration and longer sleep latency predicted worse CPAP adherence. Sleep duration mediated the association of black race with lower CPAP adherence. However, insomnia symptoms were not associated with race or CPAP adherence.

Conclusions: Among subjects with similar severity of OSA and sleepiness, baseline self-reported sleep duration and latency, but not perceived insomnia, predicted CPAP adherence over 3 months. Sleep duration explains some of the observed differences in CPAP use by race. Sleep duration and latency should be considered when evaluating poor CPAP adherence.

Portable Monitoring for Diagnosis and Management of Sleep Apnea (HomePAP)

(<http://clinicaltrials.gov/show/NCT00642486>)

NIH clinical trials registry number: NCT00642486

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Introduction

Sleep quality and quantity are associated with health outcomes; sleep duration is associated with mortality and cardiovascular morbidity.^{1,2} For example, short sleepers (less than 6 hours per night) have a greater likelihood of obesity, hypertension, diabetes, cardiovascular morbidity and mortality.³⁻⁵ Short sleepers perceive their general health status,⁶ physical and mental health to be worse.⁷ People with poor quality sleep also perceive their health to be worse and experience more physical distress.⁸ The combination of insomnia and short sleep duration in men is associated with higher mortality.⁹

Habitual sleep patterns differ by race and residential socio-economic status (SES) in large epidemiologic studies of the general population. Blacks report shorter sleep duration and longer sleep latency than whites^{10,11} yet blacks report less insomnia symptoms.¹² Residential and environmental factors may contribute to racial differences: living in poor neighborhoods is associated with shorter sleep duration.¹⁰ Neighborhood disorder (crime, noise) is associated with poor sleep quality and a lower perception of health.¹³ Perception of sleep quality differs by race and SES. Among peri-menopausal women, black women report worse sleep quality than whites and Asian women.¹⁴ Low SES is also associated with more sleep complaints.¹⁵ Therefore overall health perception is worse in populations suffering from poor quality sleep who live in poorer residential areas.

Obstructive sleep apnea (OSA) may be more severe among minorities and in those living in poorer neighborhoods, possibly due to environmental factors such as sleep disruption and air pollution.¹⁶ Continuous Positive Airway Pressure (CPAP) is an efficacious treatment for OSA,¹⁷ but CPAP adherence often limits its effectiveness.¹⁸ Prior cohort studies demonstrate that black race and low SES are associated with lower CPAP adherence.¹⁹⁻²³ Our previous analysis found a significant difference in CPAP use by race (black, non-Hispanic vs. non-

black).²⁴ Black race predicted over one-hour less use per night over three months in adjusted analyses. Though access to care, health literacy, health belief models and cost are possible reasons for these findings, few studies have explored explanatory factors. In particular, few studies have examined the relationship of habitual sleep and insomnia to CPAP use and none its association with race or SES.

Sleep apnea and insomnia frequently co-exist²⁵ and this may impact CPAP adherence. Studies show 40-50% of obstructive sleep apnea (OSA) patients have insomnia complaints.^{26,27} OSA symptoms of poor quality sleep, delayed sleep onset, fragmented sleep, and daytime sleepiness^{28,29} overlap considerably with insomnia symptoms. Subjects with chronic insomnia frequently have occult OSA.^{30,31} It is unknown why some OSA patients suffer insomnia symptoms. It is possible that demographic, cultural, environmental and socioeconomic factors contribute to symptom manifestations in OSA of poor quality sleep, insomnia and sleepiness. OSA Symptom manifestation may impact motivation as well as tolerance and subsequent adherence to CPAP therapy.

It was hypothesized that habitual sleep may differ by race and residential SES in OSA subjects and potentially serve as confounders or mediators of the association of race with CPAP use. Data from the *HomePAP* trial was used to explore the contribution of self-reported sleep measures, duration and latency, to CPAP adherence. Whether insomnia symptoms differed by demographics and were predictive of CPAP adherence was also evaluated. The goal was to identify explanatory factors for the observed differences in CPAP adherence by race.

Methods

A secondary analysis was performed on data derived from the *HomePAP* study. As described elsewhere,^{24,32} the *HomePAP* study was a randomized trial comparing unattended home-based studies to laboratory-based testing for the diagnosis and treatment of sleep apnea in subjects with moderate to severe OSA. The primary outcome for the study was CPAP adherence over the first 3 months.

Participants

The subjects included in this analysis were *HomePAP* study subjects from seven AASM-accredited sleep centers in five U.S. cities (Seattle, WA, Chicago, IL, Madison, WI, Minneapolis, MN and Cleveland, OH (the latter included 3 sites)). Subjects had a high probability of moderate to severe sleep apnea based on a clinical algorithm including neck circumference, hypertension, habitual snoring, witnessed apneas, choking or gasping. Additionally, all subjects had a minimum Epworth Sleepiness Scale (ESS) score of 12. Exclusion criteria included advanced chronic lung disease, heart failure, narcotic use or heavy alcohol use, psychiatric disorders other than mild depression, narcolepsy, severe restless legs syndrome and severe insomnia. The study protocol and procedures were approved by human subjects department and internal review boards at each participating institution.

Procedure

Eligible subjects completed questionnaires assessing demographic data, medical comorbidities, medication use and baseline sleepiness (ESS). Subjects selecting “black/African-American” and not Hispanic were classified as black. Black subjects were compared to all others (non-black) in our analysis. Level of education (>/≤ high school degree or equivalent), employment status (employed vs. unemployed) and ZIP code data were also collected.

Residential SES status (lowest quartile ZIP code SES vs. other) was derived from 2000 US census data using subjects' ZIP code at enrollment.³³ As previously described,²⁴ we created a summary ZIP code SES Z-score using multiple SES variables as in prior studies^{20,34,35}

Upon enrollment subjects completed a sleep habits questionnaire adapted from the Sleep Heart Health Study Sleep Habits Questionnaire (SHHS SHQ).³⁶ It included an evaluation of sleep duration, latency and insomnia symptoms including difficulty staying asleep, getting back to sleep and falling asleep. The SHHS SHQ has been previously validated with good reliability.³⁷ Subjects endorsing having trouble falling asleep “frequently” or “always” were characterized as having sleep-onset insomnia symptoms. Subjects characterized their typical time to fall asleep (sleep latency) in intervals (< 5 minutes, 6-30 minutes, 31- 59 minutes, 1-2 hours, over 2 hours). Sleep latency was dichotomized to \leq and $>$ 30 minutes for analyses. Subjects detailed their typical nightly sleep duration on weekends and on weekdays, responding to the question “how much time are you actually asleep” In hours and minutes. The SHQ did not specify a period of recall. Average sleep duration per night at baseline was derived from weighted averages of weekday and weekend estimates of actual nocturnal sleep times. Subjects were asked napping frequency but not duration of naps, so daytime sleep was not included in our measure of sleep duration. Sleep duration was dichotomized into short sleepers (< 6 hours) vs. others (6 hours or more).

Subjects were randomized to either home or laboratory-based diagnostic/titration studies. Subjects with an apnea hypopnea index (AHI) greater or than equal to 15 qualified for continuation in the study and CPAP titration. CPAP machines and supplies were provided at no cost to the subjects. Enrolled subjects were followed up in clinic at one and three months; their CPAP use data were downloaded. Trained staff addressed factors limiting adherence. At the

three-month follow-up subjects completed questions regarding typical sleep patterns (self-reported sleep duration) but not regarding sleep latency.

Analysis

Average sleep duration per night at baseline and three months, baseline sleep latency and sleep-onset insomnia complaints (difficulty falling asleep) were compared by race (black vs. non-black), education and ZIP code SES using the Mann-Whitney and Chi-squared tests.

Average nightly CPAP use over 3 months was compared by baseline sleep latency (\leq / $>$ 30 minutes), sleep duration ($<$ / \geq 6 hours) and sleep-onset insomnia complaints. The associations of baseline insomnia complaints, self-reported sleep duration and sleep latency with CPAP adherence were evaluated using multivariable linear regression. The association of race, education and ZIP code SES and reported sleep latency and duration was tested with logistic and linear regression.

Sleep duration and latency were assessed as mediators of the observed association of race with CPAP use, evaluating for mediation using methodology described by Baron and Kenny.^{38,39} The association of: 1. race with sleep duration and latency and 2. sleep duration and latency with CPAP adherence was determined with linear regression. The mediated effect is in essence the regression coefficient for the association of black race with sleep duration multiplied by the regression coefficient for the association of sleep duration with CPAP adherence adjusted for race (figure 1). The 95% confidence interval and p-value for the mediated effect were calculated using re-sampling methods described in MacKinnon.⁴⁰ AHI expressed as quartiles, and for study-arm (home vs. laboratory based) were controlled for in all models as these were previously identified as predictors of CPAP use in the HomePAP population.²⁴

As a sensitivity analysis, a new outcome of CPAP adherence adjusted for sleep opportunity was derived by dividing CPAP use by self-reported sleep duration at 3 months. Linear regression analyses were used to assess whether baseline sleep latency and race were predictors of percent of sleep duration using CPAP over 3 months.

Results

Of the 373 randomized and screened subjects, 191 were eligible for full study enrollment with an AHI ≥ 15 and included in analysis. Subjects were predominantly obese (87% BMI >30); 62% were white, 22% black, 9% Hispanic and 6% other; and 22% had only a high school education. The subjects reported an average nightly sleep duration of 6.3 hours (SD: ± 1.4 hours) at baseline. Sixty-six subjects (37%) reported an average of less than 6 hours of sleep and 40 subjects (22%) reported that it typically took longer than 30 minutes to fall asleep. Baseline reported sleep duration was significantly shorter in those with greater than 30 minutes sleep latency (*table 1A*). Almost one-quarter of eligible subjects (24%, $n=45$) had trouble falling asleep “frequently” to “always”. After three months of CPAP intervention, average reported sleep duration increased significantly to a mean of 6.9 (± 1.7) hours per night, $p < 0.001$.

Reported sleep measures at baseline differed by demographic factors. Black subjects reported significantly shorter baseline sleep duration than whites and Hispanics, $p=0.04$ (*table 1B*). After adjusting for education and ZIP code SES, black subjects' sleep duration was an average 43 minutes less than other groups, ($p=0.01$). Long sleep latency (> 30 minutes) was also more common among black subjects, as well as among women, subjects with only a high school or less education, and those residing in the lowest quartile SES ZIP codes (*table 1A*). High school or less education was associated with 31% greater odds of long sleep latency after adjusting for race and ZIP code SES, ($p=0.002$). Sleep-onset insomnia complaints did not correlate with reported long sleep latency.

Those with long sleep latency at baseline used CPAP less over 3 months (*table 1C*). Specifically, CPAP use averaged 67 minutes less in those with long sleep latency at baseline compared to those with shorter sleep latency ($p=0.02$); a significantly lower proportion of those

with long sleep latency used CPAP for more than 4 hours per night ($p=0.02$). Of note, CPAP use did not differ by baseline insomnia symptoms.

After controlling for AHI and study arm, baseline sleep duration and sleep latency were each predictive of 3 month CPAP use in separate linear regression models (*table 2*). One hour longer baseline sleep duration predicted 30 minutes more nightly CPAP use over 3 months. Long sleep latency predicted 70 minutes less CPAP use per night. Insomnia symptoms did not predict CPAP use nor influence the observed association between CPAP use with race, subjective sleep latency and duration.

There was some evidence that baseline sleep duration was a mediator of the association between race and CPAP use over 3 months. Table 3 shows a series of regression models that demonstrate the attenuation of the black race parameter estimate by including sleep duration. The mediated effect of sleep duration on the association of black race with CPAP use was -11.3 minutes, 95% CI (-28.0, 0.06), $p=0.05$ in formal mediation analysis (*figure 1*). In contrast, sleep latency did not appear to mediate the association of black race with CPAP use.

CPAP use as a proportion of reported sleep duration at 3 months was significantly lower in blacks than non-blacks (47% vs. 65%, $p<0.01$). In linear regression, black race and longer sleep latency remained significant predictors of the proportion of sleep duration with CPAP use (*table 4*).

Discussion

There were significant differences in CPAP adherence by black race and baseline sleep duration and latency in a racially diverse patient sample provided with comparable access to CPAP and sleep apnea management. Among the *HomePAP* study participants, self-reported longer sleep latency and shorter sleep duration at baseline were associated with black race and less CPAP use over 3 months. Through the use of statistical methods for assessing mediation, it was demonstrated that the association between black race and CPAP adherence was partially mediated by the shorter sleep duration in blacks compared to other racial/ethnic groups. Furthermore, the shorter sleep duration among blacks compared to other groups was associated with longer sleep latency. Thus, these findings point to an as of yet, under-recognized target for improving CPAP adherence: sleep duration and latency.

To our knowledge a strong association between long sleep latency and lower CPAP use, particularly in minority and low SES populations, has not been previously highlighted. Only one-third of subjects with > 30 minutes sleep latency had CPAP adherence that met Medicare requirements. After adjustment, long sleep latency predicted one hour less CPAP use nightly. Those who usually took longer to fall asleep may have had more frustration with CPAP. The face mask and air pressure, which characterize CPAP, may have been a greater nuisance to those lying awake longer. This may explain in part the reported benefit of eszopiclone on CPAP adherence in severe OSA.⁴¹ Perhaps its effects on sleep duration and latency mediate the observed greater CPAP adherence of eszopiclone users. Long sleep latency and short sleep duration may also be markers of underlying poor health or mood disorders that were not fully captured by our study measures, and these health attributes may contribute to adherence.

OSA may exacerbate insomnia and co-morbid insomnia may hinder CPAP adherence.²⁶ Insomnia symptoms have been found to be predictive of CPAP adherence in one observational

study⁴² but not in another.⁴³ In our study, subjects reporting long sleep latency did not necessarily perceive trouble sleeping and those who reported difficulties initiating sleep did not have more difficulty adhering to CPAP. This contrasts with the poor CPAP adherence among those who reported long sleep latency. The lack of association between perceived difficulty falling asleep and long sleep latency in our study may be related to our selection of sleepy subjects (ESS>12); this may have reduced the representation of individuals with the hyper-arousal insomnia phenotype. However, it is also possible that other reporting biases may have influenced our observations.

As previously noted, we observed distinct differences in sleep patterns among subgroups of our sample despite their overall similarity in OSA severity and sleepiness level. Long sleep latency appeared to be more prevalent in those from disadvantaged backgrounds with less education, unemployment and residing in lower SES ZIP codes. This is consistent with large epidemiological studies of sleep and SES: longer sleep latency was seen in those with lower employment grade (lower SES) among Japanese men.⁴⁴ Black subjects also had significantly shorter baseline sleep duration which is consistent with observational epidemiological studies of the general U.S. population.^{10,45,46} These sleep measures may be influenced by cultural patterns, health behaviors, neighborhood factors and household characteristics.⁴⁶ Long sleep latency may be a consequence of higher life stressors, psychological distress, depression and neighborhood disorder^{13,47} more common in low SES groups and minorities.

Sleep duration differed by race and explained about 12% of the observed differences in CPAP adherence by race. The current use of absolute duration of CPAP use, rather than percent of sleep duration with CPAP use, as a measure of adequate adherence may discriminate against minorities and low SES populations whose sleep duration is shorter.

However, when evaluating an alternative metric of CPAP adherence -- percent CPAP use of typical sleep duration-- which accounts for sleep opportunity, subjects of black race still had 18% less CPAP use per night of sleep. Thus, the reported differences in subjective sleep patterns do not explain all of the observed differences in CPAP use by race and unexplained determinants of CPAP adherence remain.

This secondary data analysis has several important limitations. Although objective measures of CPAP adherence were analyzed, self-reported measures of sleep latency and duration were used. These may not accurately reflect objective sleep habits. However, self-reported habitual sleep latency and duration may not differ significantly from objectively measured latency and duration.⁴⁸ We did not assess reported sleep latency after initiation of CPAP, which may better correlate with usage patterns. Also, the dataset is limited in its ability to dissect the effects of race and SES since the majority of subjects living in the lowest SES ZIP codes were black and adjustment for both race and residential SES was not feasible. Finally, the loss to follow-up was greater among the subjects in lowest SES ZIP codes and black subjects.

In conclusion, our findings highlight the need to understand sleep health disparities and consider habitual sleep latency and duration when addressing CPAP adherence particularly in those residing in poor neighborhoods and in racial/ethnic minorities. Baseline differences in sleep latency and sleep duration predicted large differences in CPAP use. Subjective sleep duration and latency differed by race, education and ZIP code SES in OSA subjects with similar levels of sleepiness and mediated some of the observed differences in CPAP use by race. Interventions to increase sleep duration and reduce sleep latency may improve CPAP adherence, especially in groups such as racial minorities who have a high prevalence of short sleep.

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Figure 1

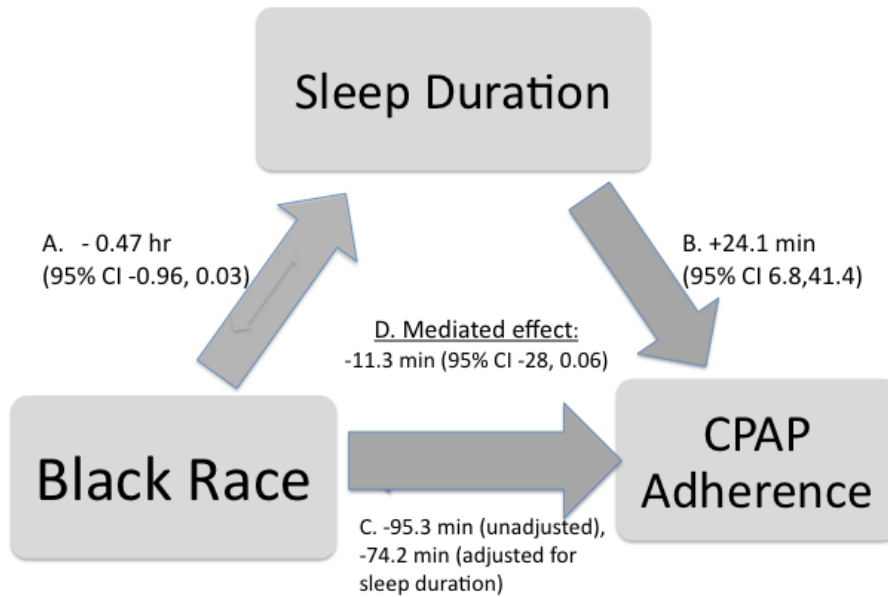


Figure 1: The mediated effect of sleep duration on the association of black race with CPAP adherence. The regression coefficients and their 95% confidence intervals (CI) are displayed below. A. Black race is associated with 0.47 hours less sleep per night. B. Each hour of sleep duration is associated with 24 minutes more CPAP use. C. The association of black race with CPAP adherence with and without adjustment for sleep duration. D. The mediated effect of sleep duration on the association of black race with CPAP adherence is 11.3 minutes. The shorter sleep duration among black subjects accounts for 11.3 minutes of the reduced CPAP use.

Table 1. Baseline characteristics of HomePAP subjects eligible for CPAP by sleep latency and sleep duration, unadjusted

Eligible participants	Total n = 191	< 30 min SL n = 144 (78%)	> 30 min SL n = 40 (22%)	p value SL	< 6 hr SDur n = 66 (37%)	> 6 hr SDur n = 115 (63%)	p value SDur
Age mean (SD)	48 (12)	49.1 (11.9)	45.4 (14.5)	0.27	48.5 (10.9)	47.6 (12.7)	0.65
Men n (%)	124 (65%)	100 (69%)	24 (60%)	0.02	57 (86%)	67 (58%)	0.58
ZIP Code SES < 25% n(%)	46 (25%)	29 (20%)	17 (42,5%)	< 0.01	16 (24%)	30 (26%)	0.57
Education n (%)^a				< 0.01			0.87
High school or less	42 (22%)	21 (14.7%)	18 (45%)		18 (27%)	20 (17%)	
> High school and < college	70 (37%)	53 (37%)	16 (40%)		44 (67%)	23 (20%)	
College or more	79 (41%)	70 (49%)	8 (20%)		25 (38%)	50 (44%)	
Work schedule n (%)^a				< 0.01			0.09
Work days	112 (61%)	95 (66%)	17 (42.5%)		43 (65%)	69 (60%)	
Evening/nights/varies	43 (24%)	32 (22%)	11 (27.5%)		26 (39%)	17 (15%)	
Unemployed	28 (15%)	16 (11%)	12 (30%)		17 (26%)	11 (10%)	
Baseline sleep traits							
Difficulty falling asleep <i>Frequently/always</i> n (%)	58 (30%)	26 (18%)	32 (80%)	< 0.01	21 (32%)	37 (32%)	0.95
ESS mean (SD)	14.3 (2.7)	14.3 (3.5)	14.3 (4.1)	0.97	14.8 (4.0)	14.0 (3.5)	0.18
SDur (hr) ^a mean (SD)	6.3 (1.4)	6.4 (1.4)	5.9 (1.3)	0.02	4.9 (0.67)	7.1 (1.0)	< 0.01
AHI mean (SD)	43.2 (26.1)	44.5 (26.4)	41.4 (26.0)	0.52	43.4 (26.1)	44.1 (26.8)	0.88

Bolded values indicate P < 0.05. Percentages reflect column percent.

^aDifferences significant by X² and Mann Whitney tests for sleep latency.

AHI, apnea-hypopnea index; CPAP, continuous positive airway pressure; ESS, Epworth Sleepiness Scale; SDur, sleep duration; SD, standard deviation; SES, socioeconomic status; SL, sleep latency.

Table 2. Baseline sleep characteristics of HomePAP subjects eligible for CPAP by race

Sleep characteristics	White (n = 119)	Black (n = 42)	Hispanic (n = 18)	p value
%Sleep latency > 30 min (n)	19% (22)	36% (14)^a	11% (2)	0.09
%Sleep duration < 6 hr (n)	33% (38)	50% (19)^a	33% (6)	0.28
Sleep duration baseline (hr) mean(SD)	6.4 (1.4)	5.9 (1.5)^a	6.4 (1.3)	0.32
Sleep duration 3 mo (hr) mean (SD)	7.0 (1.8)	6.3 (1.3)^a	6.5 (1.2)	0.26
Difficulty falling asleep baseline (% frequently-always)	24.1%	29.4%	29.4%	0.19

^aBlack versus white/Hispanic P < 0.05 in Mann-Whitney test.

CPAP, continuous positive airway pressure; SD, standard deviation.

Table 3. CPAP use at 3-mo follow-up by baseline sleep latency and duration

3 Month outcomes	Total n = 136	SL < 30 min n = 107	SL > 30 min n = 28	p value
CPAP use (min/night) ^a mean (SD)	252.8 (137)	266.8 (127.2)	199.2 (163.4)	0.02
% CPAP use of sleep duration ^a	61.6	65.4	46.9	0.01
% CPAP use > 4 hr per night ^a	56.8	60.1	44.0	0.02
% CMS CPAP compliance (> 70% nights >4 hr)	45%	48%	36%	0.26

3 Month outcomes	Total n = 136	SDur <6 hr n = 52	SDur > 6 hr n = 81	p value
CPAP use (min/night) ^a mean (SD)	252.8 (137)	218.0 (143.5)	275.9 (130.4)	0.02
% CPAP use of sleep duration	61.6	55.4	65.3	0.08
% CPAP use > 4 hr per night ^a	56.8	49.2	61.7	0.03
% CMS CPAP compliance (> 70% nights >4 hr)	45%	37%	51%	0.11

^aDifferences significant by X^2 and Mann Whitney tests, $P < 0.05$.

CMS, Centers for Medicare and Medicaid Services; CPAP, continuous positive airway pressure; SDur, sleep duration; SL, sleep latency.

Table 4. Baseline sleep latency and duration as predictors of 3 month CPAP use reported as β (standard error) in average min/day unadjusted for race

Predictor	Model 1 ^a	Model 2 ^b	Model 3 ^c
> 30 min latency	--	-70.7 (27.4)	-51.2 (27.6)
Baseline sleep duration	29.9 (8.2)	--	26.9 (8.3)
AHI II (21.0-34.5)	-7.4 (32.9)	-10.4 (33.6)	-9.5 (32.6)
AHI III (34.6-62)	40.9 (33.4)	40.1 (33.7)	39.7 (33.1)
AHI IV (> 62)	78.9 (32.6)	75.8 (33.3)	76.4 (32.3)
Study arm: home	69.9 (22.3)	71.4 (22.6)	71.4 (22.6)

^a **Model 1:** Baseline sleep duration, AHI quartile, and study arm (n =132, R² = 0.20, P < 0.001).

^b **Model 2:** Sleep latency, AHI quartile, and study arm (n =134, R² = 0.16, P < 0.001).

^c **Model 3:** Both latency and baseline sleep duration, AHI quartile, and study arm (n =132, R² = 0.18, P < 0.001).

Referent AHI: 15- 20.9

AHI, apnea-hypopnea index; CPAP, continuous positive airway pressure.

Table 5. Predictors of CPAP use at 3 mo β (standard error) in average min/day including race/ethnicity

Predictor	Model 1^a	Model 2^b	Model 3^c	Model 4^d
Black race ^e	-87.8 (31.5)	-65.0 (31.6)	-79.1 (31.2)	-60.5 (31.5)
Hispanic ^e	21.5 (37.8)	16.6 (36.1)	19.5 (37.3)	18.0 (36.6)
Baseline sleep duration	--	25.4 (8.3)	--	23.0 (8.4)
Sleep latency > 30 min	--	--	-61.1 (27.1)	-46.0 (27.4)
AHI II (21-34.5)	-3.2 (33.5)	-4.4 (32.6)	-6.2 (33.0)	-6.4 (32.4)
AHI III (34.6-62.0)	33.3 (33.6)	35.9 (33.1)	33.4 (33.1)	35.3 (32.9)
AHI IV (>62.0)	69.3 (33.3)	71.2 (32.4)	67.1 (32.8)	69.6 (32.2)
Study arm: home	73.8 (22.6)	78.9 (21.7)	75.8 (22.3)	75.5 (22.1)

^a **Model 1:** Adjusted for AHI, study arm and race/ethnicity; n =134, R² = 0.17, P < 0.001.

^b **Model 2:** Model 1 plus sleep duration; n =132, R² = 0.23, P < 0.001.

^c **Model 3:** Model 1 plus sleep latency; n =134, R² = 0.21, P < 0.001.

^d **Model 4:** Model 1 plus sleep duration and latency; n =133, R² = 0.25, P < 0.001.

^e *Referent:* white, non-Hispanic;

Referent AHI 15-20.9.

AHI, apnea-hypopnea index; CPAP, continuous positive airway pressure.

Table 6. Predictors of CPAP use per hour of sleep duration at 3 months, β (standard error) in percentage.

Predictor	Model 1 ^a	Model 2 ^b
Black race ^c	-0.17 (0.07)	-0.14 (0.07)
Hispanic ^c	0.10 (0.09)	0.09 (0.09)
Sleep latency > 30 min	----	-0.17 (0.06)
AHI ^d II (21-34.5)	0.03 (0.08)	0.02 (0.08)
AHI ^d III (34.6-62)	0.10 (0.08)	0.10 (0.08)
AHI^d IV (> 62)	0.16 (0.08)	0.15 (0.08)
Study arm: home	0.17 (0.05)	0.18 (0.05)

Bolded values indicate $p < 0.05$.

^a **Model 1:** adjusted for race/ethnicity, AHI quartile and study arm, (n =130, $R^2 = 0.16$, $P = 0.001$).

^b **Model 2:** Model 1 plus baseline sleep latency, (n =130, $R^2 = 0.21$, $P < 0.001$).

^c *Referent:* white, non-Hispanic.

^d *Referent* AHI 15-20.9,

AHI, apnea-hypopnea index; CPAP, continuous positive airway pressure.

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